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The Accuracy Medication to Treat Urothelial Carcinoma

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INTRODUCTION

The treatment of urothelial carcinoma is challenging. While known therapies are effective, the results can be variable. Known treatment modalities include transurethral resection of the bladder tumor, intravesicular BCG, chemotherapy, immune check point inhibitors, and antibody drug conjugates. Finding certain patient and tumor characteristics to determine responders to therapy can help personalize the treatment approach and optimize results.

DESCRIPTION

Precision medicine offers a solution to this problem by providing clinicians with tools such as liquid biopsies, prognostication models, and biomarkers to identify essential patient characteristics. Furthermore, precision medicine improves treatment efficacy by identifying and exploiting specific targets. In this review, we discuss available tools in precision medicine, describe ongoing clinical trials, and identify areas for future study. Since the advent of the Precision Medicine Initiative in 2015, efforts have been made to develop personalized diagnostic and therapeutic approaches to cancer care that take into account individual differences including somatic genomic, transcriptomic, and proteomic alterations, as well as germline genetic mutations. Significant advancements have been made that have changed the therapeutic landscape of advanced urothelial carcinoma, including the emergence of immune checkpoint inhibitors (PD1/L1 inhibitors), antibody drug conjugates, and a targeted agent. Moreover, the application of PD1/L1 inhibitors to earlier disease settings has improved outcomes, e.g., muscle-invasive and non-muscle invasive bladder. The inter and intra-tumor heterogeneity of bladder cancer has been well documented and is a challenge in delivering precise and efficacious treatment. “Liquid biopsies” which utilize peripheral blood to assess for circulating tumor cells (CTCs) and circulating tumor DNA (ctDNA) have gained significant attention due to their non-invasive nature and potential in identifying driver mutations. CTCs are thought to originate in the primary tumor and enter circulation as they metastasize to distant organs, while ctDNA includes DNA mutations, epigenetic alterations, and other forms of tumor-specific abnormalities. Vandekerkhove et al. demonstrated a mutational concordance of 83.4% between ctDNA and matched bladder tumor tissues. In addition, 90% of mutations remained consistent across serial ctDNA samples, while concordance for serial tumor tissue was significantly lower, indicating that the identification of driver mutations might be better identified in plasma. CTCs have also shown potential in predicting therapeutic response and prognosis. Soave et al. demonstrated that the presence of CTCs is associated with increased disease recurrence, cancer-specific mortality, and overall mortality in patients who did not receive adjuvant chemotherapy prior to radical cystectomy in patients with recurrent Ta, T1, CIS refractory to TURBT, or muscle-invasive bladder cancer (MIBC). In patients with CTCs who received adjuvant chemotherapy, there was no difference in disease recurrence and cancer-specific or overall mortality. The presence of CTCs may provide guidance on whether to treat UC with adjuvant chemotherapy; however, further investigation is required before this can be clinically applied. Another study demonstrated that quantifying CTCs may also predict prognosis. Alva et al. collected blood samples from 20 patients with BC who were eligible for neoadjuvant chemotherapy and observed that patients with medium to high tumor cell levels at baseline and follow-up had an unfavorable pathological stage disease.

CONCLUSION

However, as outlined above, there is still much room for improving treatment efficacy and predicting prognosis and treatment response. Moreover, the prediction of severe toxicities needs greater focus to optimize the therapeutic index of anti-cancer therapy.

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